

# INTERVENTIONAL NEURORADIOLOGY

## Endovascular Therapy of the Central Nervous System

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## CHAPTER 5

# Embolization of Intracranial Aneurysms with Detachable Coils and Electrothrombosis

Guido Guglielmi

Approximately 2% of the entire population harbors an intracranial aneurysm. Twenty-five thousand intracranial aneurysms rupture in North Americans every year, leading to bleeding into the subarachnoid space or intracerebral tissue (1). The primary purpose of the treatment of a ruptured aneurysm is to prevent a catastrophic, and often fatal, rebleeding. The aneurysm should be occluded as soon as possible after the hemorrhage (24–48 hr), to avoid a second hemorrhage and to aggressively manage post-hemorrhagic arterial vasospasm (2,3). Microsurgical clipping of the neck of the aneurysm, with preservation of the parent artery, is the current treatment of choice for this disease. This therapeutic modality requires general anesthesia, open craniotomy, brain retraction, and manipulation in order to reach the neck of the aneurysm.

The capacity of intracranial, intra-arterial navigation has opened a new modality of therapy for intracranial aneurysms. This type of therapy uses the arteries as natural channels to reach the neck and sac of the aneurysm, without the need of a craniotomy. The present technology produces soft microcatheters that allow a safe and less traumatic intra-arterial navigation into an aneurysm or into its parent artery, very close to its neck.

Intravascular embolization of aneurysms may be divided into two broad categories, depending upon their size and location: (i) intravascular occlusion of the aneurysm and parent artery and (ii) intravascular occlusion of the aneurysm, with preservation of its parent artery.

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### INTRAVASCULAR ANEURYSM OCCLUSION WITH SACRIFICE OF PARENT ARTERY

This therapeutic modality is mainly utilized for the management of internal carotid, intracavernous, and supraclinoid giant aneurysms, as well as posterior fossa unclippable aneurysms.

Giant aneurysms (diameter larger than 25 mm) usually present with clinical evidence of intracranial mass effect, though they also may hemorrhage. The presence of partial thrombosis of its lumen or calcification in its wall does not preclude the possibility of a severe intracranial hemorrhage. These aneurysms are classically difficult to treat by surgical techniques because their large size limits both (a) the intraoperative manipulation of the sac of the aneurysm and (b) a clear dissection of an unusually wide neck (4).

Unruptured giant intracavernous aneurysms present with a progressive cavernous sinus syndrome manifested by third, fourth, and sixth cranial nerve palsies and retro-orbital pain related to compression of the fifth nerve. An intracavernous aneurysm may rarely rupture, producing a spontaneous high-flow, carotid-cavernous fistula. If the dome of the aneurysm protrudes into the subarachnoid space, this may also be the origin of a intracranial hemorrhage.

Serbinenko (5) and Debrun et al. (6–8) pioneered the use of detachable balloons to treat unclippable intracavernous aneurysms. In 1984, Berenstein et al. (9) reported their experience in the use of detachable aneurysms for therapy of nine carotid-cavernous aneurysms. Their technique included trapping of the aneurysm by detaching a balloon distal to, and another balloon proximal to, the neck of the aneurysm. They stressed the importance of pre-embolization clinical neurological tolerance by performing temporary balloon occlusion of the internal carotid artery (ICA) with a double-lumen-

balloon catheter with the patient under systemic heparinization.

Fox et al. (10) in 1987 and Higashida et al. (11) in 1990 reported their casuistics in 37 and 68 intracavernous aneurysms, respectively. Both authors performed permanent occlusion of the ICA via a transfemoral, endovascular approach. Their protocols included: systemic heparinization; transfemoral technique; and neurological tolerance test with temporary balloon occlusion of the ICA for 20–30 min, followed by permanent occlusion of ICA with a contrast-filled balloon located proximal to or across the neck of the aneurysm. A second balloon was then detached proximal to the first one in order to decrease the possibility of early deflation and untoward intracranial migration of the first balloon. Fox et al. (10) used latex balloons, whereas Higashida et al. (11) preferred silastic balloons.

Fox et al. (10) reported complete aneurysm obliteration in all 37 intracavernous aneurysms, after proximal balloon occlusion of the ICA. In 11 patients (29.7%), an extracranial-to-intracranial (EC-IC) bypass was performed before the ICA occlusion. Three of those 11 patients developed postembolization transient ischemic complications, and two had temporary worsening of their cranial nerve palsies.

Higashida et al. (11) also reported complete aneurysm obliteration in 68 giant intracavernous aneurysms, using the endovascular technique already described. Two patients had a pre-embolization EC-IC bypass because they developed neurological deficit during the ICA tem-

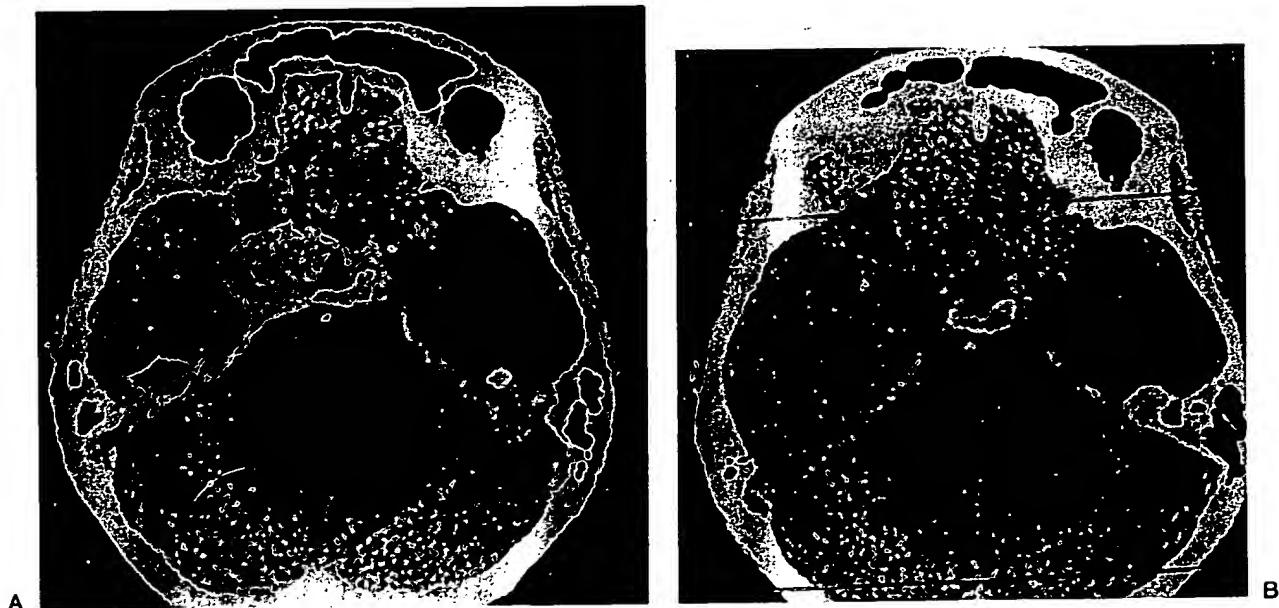
porary balloon occlusion. Seven patients developed postembolization transient brain ischemia. Three patients had a permanent stroke following the balloon ICA occlusion.

Fox et al. (10) also treated 21 unclippable supraclinoid aneurysms using a similar technique. They obtained a complete aneurysm obliteration in 10 cases (47.6%). In some of the remaining 11 patients a surgical intracranial trapping procedure was performed in order to isolate the residual aneurysm from the circle of Willis or from the ophthalmic artery. Four patients developed postembolization transient brain ischemic complications.

Berenstein et al. (9), Fox et al. (10), Serbinenko (5,12), Romodanov and Shcheglov (13), Higashida et al. (14), and Aymard et al. (15) have reported balloon therapy of vertebrobasilar aneurysms with balloon occlusion of one or both vertebral arteries and sometimes of the trunk of the basilar artery (14).

Aymard et al. (15) treated 21 patients with inoperable vertebrobasilar aneurysms by balloon-occluding one or both vertebral arteries in the neck. They achieved a complete angiographic and clinical cure in 13 cases (61.9%). A partial aneurysm thrombosis was observed in six cases (28.6). One patient developed a transient Wallenberg syndrome, and two patients died (9.5%).

In summary, the permanent balloon occlusion of a parent artery may be successful in a wide variety of intracranial, giant unclippable aneurysms. It is believed that the diminished pulsations of the giant thrombosed aneurysms, combined with the progressive retraction of the



**FIG. 1. A:** Contrast-enhanced computerized tomogram shows a giant, partially thrombosed, right carotid-cavernous aneurysm. **B:** Contrast-enhanced computerized tomogram performed 4 months after permanent right internal carotid artery balloon occlusion. The aneurysm is no longer visible.

intra-aneurysmal clot, produce clinical improvement due to noticeable decrease in the mass effect of the aneurysm (Fig. 1).

Endovascular balloon obliteration of the parent artery may have some advantages over its surgical occlusion because it may be performed with the patient awake, thereby allowing functional neurological and electroencephalographic monitoring during temporary balloon occlusion of the parent artery. The contralateral carotid and vertebral circulation can be injected during the temporary balloon occlusion test in order to verify if the aneurysm has been effectively isolated from the circle of Willis or from the ophthalmic artery. It needs to be said that a negative functional test during temporary balloon occlusion of the parent artery does not completely exclude a delayed neurological deficit (10). This clinical complication may be seen in approximately 4% of all cases of parent artery occlusion, and it may be related to untoward distal embolization from clots arising from a partially thrombosed aneurysm or from an episode of prolonged hypotension in patients with compromised brain collateral circulation.

#### INTRAVASCULAR OCCLUSION OF ANEURYSMS WITH PRESERVATION OF THE PARENT ARTERY

##### Balloon Embolization

The present technology allows endovascular occlusion of aneurysms with preservation of the parent artery. In 1974, Serbinenko (5) reported partial occlusion of anterior and posterior circulation aneurysms using detachable latex balloons. In 1978, Debrun et al. (7) occluded five intracavernous aneurysms, preserving the ICA lumen. Follow-up angiograms demonstrated anatomical reconstitution of most of the aneurysms, in spite of the fact that they filled the balloons with silicone.

In 1982, Romodanov and Shcheglov (13) reported balloon occlusion of 119 anterior-circulation aneurysms and of one posterior-circulation aneurysm. In 93 cases it was possible to preserve the lumen of the parent artery (78%). These authors punctured the ICA in the neck and manipulated detachable latex balloons. They also filled the balloon with silicone before detaching it, to avoid balloon deflation and reconstitution of the aneurysm. They reported two deaths in these 93 patients; one of them was related to subarachnoid hemorrhage (SAH) due to aneurysm rupture, and the other was related to massive intracranial embolization with semisolid silicone due to rupture of the balloon. The aneurysm could not be occluded in 11 patients (9.2%). Three of these patients had fatal complications during embolization. In 15 patients (12.6%), the aneurysm was excluded from

the circulation by occluding the parent artery. Three of these patients had fatal neurological complications related to ICA occlusion. Romodanov and Shcheglov (13) state that balloon embolization of aneurysms is contraindicated in intracranial hematoma, in small aneurysms, in aneurysms with a wide neck, in the acute phase of SAH, and in the presence of severe arterial vasospasm.

In 1981, Hieshima et al. (16) described the use of silastic balloons for the endovascular occlusion of intracranial aneurysms. They reported 84 cases of nonoperable, high-risk anterior-circulation (59 cases) and posterior-circulation (25 cases) aneurysms in which they embolized the aneurysm while preserving the parent artery (36). The silastic balloon is solidified with hydroxyethyl methacrylate (HEMA) before being gently detached from the microcatheter. The aneurysm was completely occluded in 65 cases (77.4%) and almost completely obliterated in 19 cases (22.6%). They reported 15 deaths (17.9%) and nine neurological complications (10.7%) related to therapy. Their high morbidity and mortality rates may be explained by the type of aneurysms they treated (nonclippable aneurysms located in very difficult anatomic areas) and by the patients' poor medical condition.

The critical analysis of the available data on balloon occlusion of aneurysms shows that this technique is mostly applied in (a) symptomatic, unruptured, unclippable aneurysms (intracavernous aneurysms, giant aneurysms) and (b) symptomatic, ruptured, unclippable aneurysms due to location or patients' medical conditions.

Due to the fact that unruptured symptomatic aneurysms are a minority group (6.8%) among all treated aneurysms (17), that 98.4% of all anterior circulation aneurysms are amenable to surgical therapy, and that aneurysm treatment needs to be performed in their acute phase (2,3) to improve clinical results, the balloon treatment of aneurysms has still played a minor role in the therapeutic management of all intracranial aneurysms.

##### Embolization of Aneurysms with Coils

Avoiding craniotomy, taking advantage of the fact that arteries are natural channels through which aneurysms can be reached, has led some researchers to find the way of treating cerebral aneurysms via endovascular approach. The endovascular approach to aneurysm occlusion previously has been limited to balloon embolization. Balloon embolization has the special advantage of avoiding general anesthesia, craniotomy, and brain manipulation. However, the main disadvantage is that the fragile wall of the aneurysm undergoes stress while adapting to the shape of the balloon, thus carrying the risk of aneurysm rupture. This risk may be higher in the acute post-hemorrhagic phase.

A new technique of intravascular embolization of intracranial aneurysms using detachable coils has been developed in an attempt to improve anatomical and clinical results (18,19). The embolic material is a soft, detachable platinum coil, 4–40 cm in length, soldered to a stainless steel delivery wire. The tip of a microcatheter is positioned in the aneurysm, using the transfemoral approach. A detachable platinum coil is then advanced through the microcatheter and is positioned in the aneurysm. A positive electric charge of 0.5 mA is applied to the proximal end of the detachable coil, thus producing attraction of the negatively charged red blood cells, white blood cells, platelets, and fibrinogen around the platinum coil (see section entitled "Electrothrombosis," below). The positive electric current also dissolves the stainless steel immediately proximal to the junction between the stainless steel and the platinum coil, detaching the coil within the aneurysm (see section entitled "Electrolysis," below). Numerous coils may be deposited in an aneurysm, using this technique. The aneurysm is slowly and gently filled with coils until it is completely and tightly packed. The detachable coils adapt themselves to the shape of the aneurysm, and they appear to produce much less deforming pressure upon the wall of the aneurysm than does an inflated silastic or latex balloon.

#### **Background and History**

Electrothrombosis consists of the production of an endovascular thrombus by a positively charged endovascular metallic electrode. Velpau (20) in 1831 and Phillips (21) in 1832 were the first to introduce needles into the lumen of a vessel, producing an intraluminal clot and withdrawing the needle after the thrombus had formed. The same authors suggested that the introduction of metals with high dissociation constant of positively charged ions ( $\text{Fe}^{2+}$ ,  $\text{Cu}^{2+}$ , and  $\text{Be}^{2+}$ ) might be useful in the treatment of aneurysms. In 1864, Moore and Murdoch (22) were the first to introduce a permanent wire into the sac of an aortic aneurysm in order to produce an intra-aneurysmal thrombus. In 1847, Ciniselli (23) was the first to apply a positive electric current, passing it through needles temporarily positioned into the sac of an aneurysm. In 1952, Bigelow and De Foyes (24) demonstrated that washed platelets migrated to the positive pole of an electrophoretic cell. Abramson (25) had demonstrated earlier that a similar phenomenon occurred with white blood cells. In 1953, Sawyer and Pate (26) showed that the use of direct electric current through citrated or heparinized blood led to precipitation of blood elements around the positively charged electrode (anode). These elements were red blood cells, white blood cells, platelets, and fibrinogen. They used a 0.2- to 10-mA current for approximately 30 min.

#### **Electrolysis**

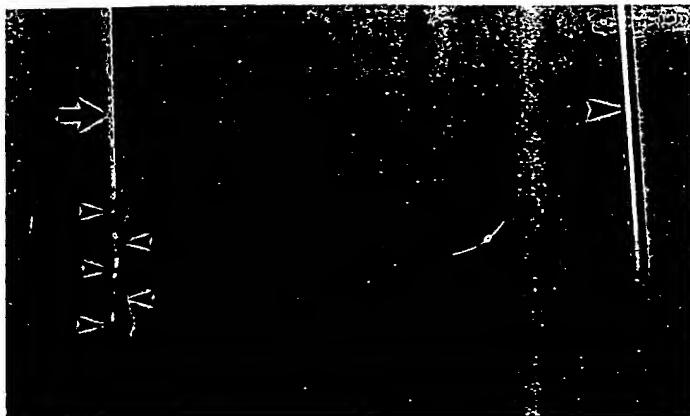
Miller et al. (27) utilized 5- to 10-mA direct electric current to produce a thrombus in the citrated blood of dogs; the electrodes were made of either stainless steel or platinum. These authors proved that platinum is three to four times more thrombogenic than stainless steel, that stainless steel dissolves during the passage of current by electrolysis, and that there is no difference in the size of the clot if the diameter of the platinum electrode is changed from 0.25 to 0.9 mm. They also observed that the size of the clot was directly proportional to the time of the application of the current. Piton et al. (28) tested various metals in saline. This included silver, copper, platinum, and stainless steel electrodes, all 0.6 mm in diameter. They applied a 10-mA direct electric current to these electrodes and showed that the silver electrode underwent electrolysis in 22 min and that the stainless steel electrode did so in 12 min; the copper electrode was rapidly affected by oxidation, and the platinum was not electrolyzed.

#### **Polarity of the Vessel Wall**

In 1953, Sawyer and co-workers (26,29,30) performed a series of experiments in dogs and showed that the intima of vessels has a constant negative charge (between –3 and –15 mV) when compared to the adventitia. Because of this negative charge the negatively charged blood elements are normally repelled from the intima, decreasing the possibility of organization of an intravascular thrombus. If the intima is injured, immediate reversal of the electrical polarity takes place so that it becomes positively charged, attracting the negatively charged blood elements. This phenomenon may play an important role in post-traumatic vascular clotting.

#### **Electrothrombosis (Fig. 2)**

In 1961, Salazar (31) produced complete thrombosis of the coronary arteries in dogs by applying a very small intravascular positive electric current (0.5 mA, 3 V) for 2 hr. Araki et al. (32) studied electrical thrombosis by applying a 3-mA positive direct electric current for 1 hr. Using this technique, they showed that a thrombus could be formed in the carotid artery of dogs in 90% of cases. Thrombus formation was reduced if the artery was infused with heparin. Subsequently, Piton et al. (28) elicited electrothrombosis of the aorta, femoral artery, and common carotid artery in rabbits by applying electric current (10 mA, 9 V) to a platinum or stainless steel endovascular electrode (anode). Guglielmi et al. (33) reported intra-aneurysmal thrombus formation in saccular



**FIG. 2.** Electrothrombosis is tested via an *in vitro* experiment. Two stainless steel 0.038-in. guidewires (arrow and arrowhead) were immersed simultaneously in a metallic cup containing heparinized blood. A 0.5-mA positive direct electric current was passed through the guidewire on the left (arrow) for 10 min, whereas no current was passed through the other guidewire. At the end of the experiment, a thrombus (arrowheads) was seen covering the guidewire through which the current had been passed, whereas none was seen around the other guidewire.

lar aneurysms created on the common carotid artery of 10 rabbits. Using an endovascular approach, they introduced a stainless steel electrode through a transfemoral microcatheter into the aneurysms and applied a 10-mA positive direct electric current to initiate intraneurysmal thrombosis.

Based upon the *in vitro* and *in vivo* experiments described above, it is possible to say the following: (a) If a positive direct electric current is used within a vessel, a thrombus will precipitate on the positive electrode (anode); (b) the size and weight of thrombus is directly proportional to the coulombs (milliamperes  $\times$  minutes) of electricity delivered; (c) platinum appeared to produce the largest clots and is not affected by electrolysis; and (d) stainless steel electrodes are electrolyzed in a few minutes.

#### *Clinical Application of Electrothrombosis: Extravascular Approach*

Mullan (34) was the first to use electrothrombosis to occlude saccular aneurysms in a large number of patients. His technique consisted of stereotactic insertion through a burr hole of fine copper-plated steel needles, 0.2 mm in diameter, across the neck of the aneurysm at 1.0-mm intervals. Thrombosis was initiated by passing a positive direct electric current through each needle for 5 min. Postembolization angiograms were obtained every 30 min until satisfactory thrombosis was achieved. With this technique, a satisfactory occlusion of the aneurysm was achieved in 49 patients; an incomplete occlusion resulted in eight patients, all of whom died 1–66 days following embolization due to recurrent intracranial hemorrhage. Mullan concluded that stereotactic copper electrothrombosis offered a risk comparable to that associated with conservative management followed by surgical clipping. This technique has not become very popular because the aneurysm has to be punctured, extensive

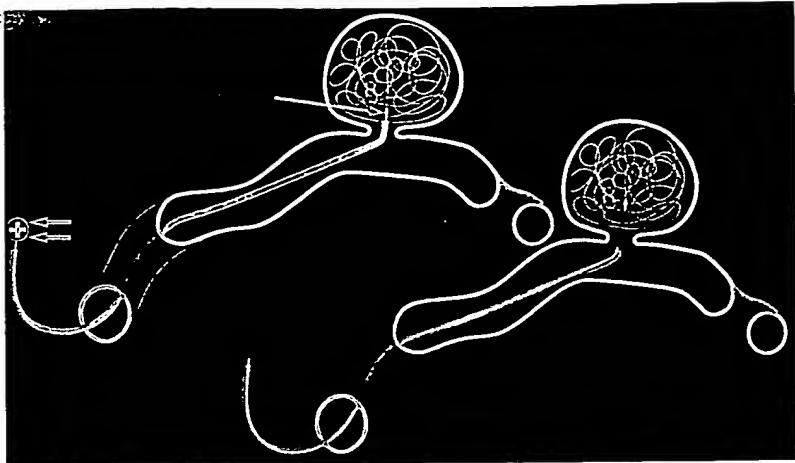
equipment is required, it is necessary to penetrate cerebral tissue in order to reach the aneurysm with the probe, and it is unsuitable for a wide spectrum of aneurysms.

#### *Clinical Application of Electrothrombosis: Endovascular Approach*

Thirty-nine patients with intracranial saccular aneurysms which were difficult to treat surgically were treated via endovascular approach between March 1990 and August 1991. Twenty-one of these were women and 12 were men. The patients ranged in age from 21 to 73 (mean age: 49). Clinical presentation of 18 of the patients was SAH, 11 presented with mass effect, and two patients presented with other symptoms. In two other patients the aneurysm was discovered incidentally. All but four patients presenting with SAH were in grade I-II—those four were in grade IV-V (35). Eleven aneurysms involved the basilar bifurcation, four involved the intracavernous carotid, and three involved the vertebrobasilar junction. The remaining 13 aneurysms involved the following: the posterior inferior cerebellar artery; the internal carotid artery bifurcation; the anterior communicating artery; and the trigeminal, carotidophthalmic, middle cerebral, superior cerebellar, and posterior communicating arteries. Of all the aneurysms treated, 18 were small (<12 mm), 9 were large (12–25 mm), and 12 were giant (>25 mm). (See Table 1).

#### *Occlusion Technique*

All procedures are performed with the patient awake and under systemic heparinization (3000 IU at the beginning and subsequently 1000 IU per hour). The transfemoral approach and digital subtraction angiography with "road-mapping" capability are always used. The proximal parent artery is catheterized with a No. 6 non-tapered polyethylene guiding catheter. A Tracker-GDC



**FIG. 3.** Steps in electrothrombosis and electrolysis of saccular aneurysms via endovascular approach are represented in this diagram. Upper left: Introduction of the platinum distal portion of a stainless steel delivery wire into the aneurysmal sac through a microcatheter. Arrow points to the junction between the platinum and the stainless steel. Positive direct electric current is applied to the guidewire (double arrows) so that the positively charged platinum attracts the negatively charged blood components, thus initiating thrombus formation. Lower right: In 4–12 min the current has also dissolved (by electrolysis) the stainless steel portion proximal to the junction; in addition, the platinum portion is detached within the clotted aneurysm.

microcatheter is then advanced coaxially into the aneurysm sac with the aid of a microguidewire (Tracker-10 GDC, Tracker-18 GDC, Seeker-10 Lite, Seeker 14; Target Therapeutics, San Jose, CA). In case of lateral aneurysms (i.e., aneurysms arising at a 90° angle with the parent vessel), it is necessary to curve the tip of the microcatheter with steam. The Tracker-GDC microcatheter has two radiopaque markers: One is at the distal end, and the other is 3 cm proximal to the first one. The guidewire is removed once the microcatheter tip is inside the aneurysm sac in order to avoid aneurysm perforation. Continuous pressurized flushing with heparinized saline of both coaxial systems is utilized during the whole procedure to decrease friction, to eliminate the possibility of clot formation, and to avoid electric current dispersion. An intra-aneurysmal angiogram may be performed to make sure that the microcatheter is positioned properly. The detachable coil (GDC, Target Therapeutics, San Jose, CA) is then introduced into the microcatheter with the aid of a special introducer.

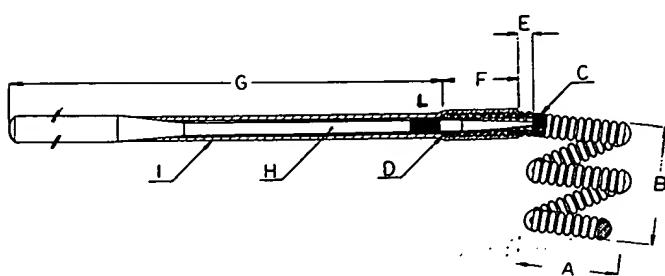
#### Detachable Coils

The “detachable coils” can be 0.010 or 0.015 in. in diameter (this 50% increment in diameter leads to a 100% increment in volume). The former is used for small aneurysms and is compatible with the Tracker-10

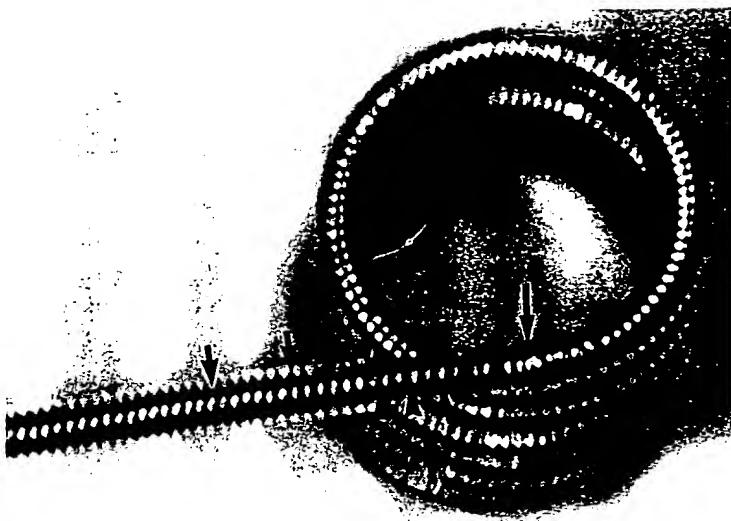
GDC microcatheter. The detachable coil has four components (Fig. 4): The proximal part (175 cm in length) is made of stainless steel core wire, the intermediate platinum portion is a 5-mm-long platinum marker, the intermediate stainless steel portion (3 cm in length) is made of a very soft stainless steel coil, and the distal component varies from 4 to 40 cm and is a coil made of platinum. It has a circular memory that allows its deposit in the dome of the aneurysm without traumatizing its fragile walls.

The distal 30 cm of the proximal portion, the platinum marker, and most of the intermediate part of the stainless steel delivery system are laminated with Teflon to achieve electrical insulation. The distal 2-mm portion of the stainless steel coiled portion is uninsulated to allow electrolytic detachment (Fig. 5).

Currently, eight types of coils are available and are selected depending upon the aneurysm sac and neck size. They are: (i) an 8-cm-long helix with a 2-mm circular memory, (ii) a 10-cm-long coil with a 4-mm circular memory, (iii) a 15-cm-long coil with a 5-mm circular memory, (iv) a 20-cm-long coil with a 6-mm circular memory, (v) a 20-cm-long coil with an 8-mm circular memory, (vi) a 40-cm-long coil with an 8-mm circular memory, (vii) a 20-cm-long coil (0.015 in. in diameter) with an 8-mm circular memory, and (viii) a 40-cm-long coil (0.015 in. in diameter) with an 8-mm circular memory (Fig. 6).



**FIG. 4.** Diagram of the detachable platinum coil. A: diameter of the circular memory. B: The distal portion (10–40 cm in length) made of coiled platinum wire. C: Microsolder connecting the distal portion to the intermediate stainless steel portion. D: Microsolder connecting the intermediate stainless steel portion to the intermediate platinum portion. E, F: The intermediate stainless steel portion (3 cm in length) consisting of coiled stainless steel wire. E: The insulated part of the intermediate stainless steel portion. G: The proximal stainless steel portion (175 cm in length). H: The tapering stainless steel core wire. I: Teflon lamination. L: The intermediate platinum portion is a marker (0.5 cm in length) consisting of coiled platinum wire.



**FIG. 5.** Platinum spiral with circular memory. The microsolder that connects platinum to stainless steel is visible (*longer arrow*). The part that is dissolved by electrolysis (the uninsulated stainless steel coil, 2 mm in length) is shown (*arrowheads*). The opalescent Teflon lamination is also visible (*shorter arrows*).

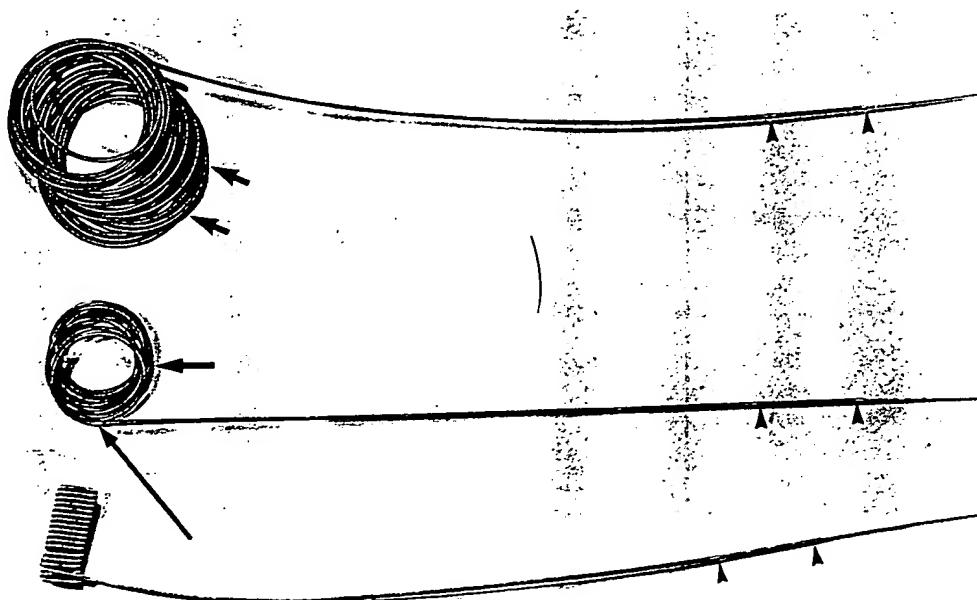
#### *Delivery of the Detachable Coils*

When the platinum coil is positioned in the microcatheter, it adopts a straight shape that allows it to be advanced toward the aneurysm without significant friction. As soon as the platinum coil emerges from the microcatheter, it adopts a circular formation and folds upon itself, decreasing the possibility of migration outside aneurysmal neck and conforming to the shape of the aneurysm without causing aneurysmal wall distortion.

Platinum is more radiopaque than stainless steel, so that the platinum distal component is easy to see under

fluoroscopy. This property allows control of the coil deposited in the aneurysm and ensures that none is placed in the parent vessel. When such a migration was observed, the coil was withdrawn and repositioned inside the aneurysm or exchanged with a different-sized one. This maneuver is performed by pulling back the delivery guidewire and advancing it again through the microcatheter.

When the detachable platinum coil is seen to be in suitable position inside the aneurysm and with the platinum/stainless steel junction zone 3 mm beyond the tip of the microcatheter, intra-aneurysmal electrothrombo-



**FIG. 6.** Various detachable platinum coils used for aneurysm occlusion: a 40-cm-long platinum coil with an 8-mm circular memory (*short arrows*); a 15-cm-long platinum coil with a 6-mm circular memory (*medium-sized arrow*). The solder between the platinum and the stainless steel components of the detachable coil is shown (*long arrow*). The platinum marker, 0.5 cm in length, is also visible (*arrowheads*).

TABLE 1. Profile of 39 patients treated with a GDC aneurysm occlusion system\*

Case no.	Age (years)	Sex	Clinical presentation	Aneurysm location	Aneurysm size <sup>b</sup>	Neck size <sup>c</sup>	Coils length	Result (% occlusion)	Complications related to technique	Outcome
1	28	F	CC fist.	Cavernous	Small	Small	35 cm	Fistula cured	None	Good
2	21	M	CC fist.	Cavernous	Large	Small	20 cm	Fistula cured	None	Good
3	33	F	SAH	PICA	Small	Small	16 cm	80%	None	Good (surgery)
4	60	F	Mass	Bas. bif.	Giant	Wide	265 cm	80%	None	Good
5	45	M	Mass	ICA bif.	Large	Wide	80 cm	70%	Transient aphasia resolved in 2 days	Good
6	58	M	SAH	VB junct.	Large	Wide	140 cm	95%	None	Good
7	46	M	SAH	Bas. bif.	Large	Wide	120 cm	80%	None	Good
8	47	F	SAH	Car. ophth.	Giant	Wide	55 cm	70%	None	Good
9	58	F	Mass	Cavernous	Giant	Wide	120 cm	Cured	Incomplete coil delivery; balloon ICA occlusion	Good
10	56	F	SAH	Bas. bif.	Small	Wide	168 cm	95% (three Embos)	None	Good (surgery)
11	50	M	SAH	ACoA	Small	Wide	38 cm	90%	None	Good
12	43	F	Mass	Trig. art.	Giant	Wide	220 cm	70%	None	Good
13	69	F	SAH	Bas. bif.	Small	Wide	15 cm	95%	None	Good
14	67	F	Mass	MCA	Giant	Wide	384 cm	98% (three Embos)	None	Good
15	57	F	SAH	Bas. bif.	Small	Small	55 cm	100%	None	Good
16	32	M	SAH	PCoA	Large	Wide	360 cm	80%	None	Good (surgery)
17	45	F	Mass	Bas. bif.	Giant	Wide	155 cm (0.015 in.)	90%	None	Good
18	51	F	Incid. finding	Paraphth.	Small	Wide	15 cm	Cured	Incomplete coil delivery; balloon ICA occlusion	Good
19	48	F	SAH (grade V)	ICA bif.	Small	Small	95 cm	100%	None	Dead (grade V)
20	70	F	SAH	SCA	Small	Wide	115 cm	90%	None	Good
21	56	M	SAH	Bas. bif.	Small	Wide	120 cm	100%	Thrombus progression resulting in PCAs occlusion	Hemianopia

22	29	F	Incid. finding	Bas. bif.	Small	23 cm	100%	NA	Good	None
23	67	F	SAH (grade V)	PICA	Small	8 cm			Aneurysm rupture with microcatheter after first coil delivery	Dead (grade V)
24	32	F	SAH	Car. ophth.	Small	125 cm	100%		None	Good
25	34	M	Mass	Bas. bif.	Wide	200 cm (0.015 in.)	90%		None	Good
26	42	F	SAH	VB junct.	Small	14 cm	95%		None	Good
27	70	M	Mass	Bas. bif.	Giant	53 cm	95%		None	Good
28	56	F	Mass	Bas. bif.	Large	255 cm	98%		None	Good
29	44	M	SAH (grade V)	MCA	Small	70 cm	100%		None	Dead (grade V)
30	38	M	Mass	VB junct.	Wide	400 cm (0.015 in.)	85%		None	Good
31	73	F	Mass	Cavernous	Giant	200 cm (0.015 in.)	90%		None	Good
32	61	M	SAH	ICA bif.	Giant	326 cm (0.015 in.)	95%		None	Good
33	30	F	SAH	Car. ophth.	Small	68 cm	100%		None	Good
34	40	F	Incid. find.	Car. ophth.	Large	140 cm (0.01/0.015 in.)	90%		None	Good
35	45	F	Intrav. hemorrh.	Bas. bif.	Small	93 cm	100%		None	Dead (CSF infection)
			(grade IV)							
36	48	F	Mass	Car. ophth.	Giant	1420 cm (0.015 in.)	98%		Good	
37	56	F	Mass	Bas. bif.	Small	150 cm (0.01/0.015 in.)	100%		Good	
38	72	F	Mass	Car. ophth.	Giant	118 cm (0.01/0.015)	100%		Good	
39	48	F	Incid. find.	Car. ophth.	Large	75 cm (0.01/0.015 in.)	95%		Good	

\* ACoA, anterior communicating artery; Bas. bif., basilar bifurcation; Car. ophth., carotid ophthalmic; CC fist., carotid-cavernous fistula; CSF, cerebrospinal fluid; Embos, embolizations; ICA, internal carotid artery; ICA bif., internal carotid artery bifurcation; Incid. find., incidental finding; Intrav. hemorrh., intraventricular hemorrhage; Mass, mass effect; MCA, middle cerebral artery; NA, not available; Paraophth., paraophthalmic artery; PCA, posterior cerebral artery; PCoA, posterior communicating artery; PICA, posterior inferior cerebellar artery; SAH, subarachnoid hemorrhage; SCA, superior cerebellar artery; Trig. art., trigeminal artery; VB junct., vertebrobasilar junction.

<sup>a</sup> Small: <12 mm. Large: 12–25 mm. Giant: >25 mm.

<sup>b</sup> Small: <4 mm. Wide: >4 mm.

<sup>c</sup> Small: <4 mm. Wide: >4 mm.

sis and electrolysis are generated by applying a 0.5/0.7-mA, 2.2/2.9-V positive direct electric current to the proximal end of the stainless steel guidewire using a battery-operated current generator. The negative ground pole is connected to a surface or needle electrode at the groin. In this way the intra-aneurysmal platinum becomes positively charged and attracts the negatively charged white blood cells, red blood cells, platelets, and fibrinogen, thus electrically inducing thrombus formation (Fig. 7).

By the time a thrombus forms around the platinum coil, the current has also dissolved the uninsulated intra-aneurysmal stainless steel coil closest to the platinum coil by electrolysis (Fig. 7). The detachment of the coil occurs within 4–12 min. By taking advantage of this phenomenon, it is possible to detach the platinum coil within the aneurysm without any need for pulling. In order to detect the detachment, the generator has a milliamperemeter and a voltmeter. A sudden drop in current or a sudden raise in voltage indicates that detachment has occurred (i.e., the electrical resistance has increased). The stainless steel delivery wire is then withdrawn.

The platinum coils represent a packing material that holds the intra-aneurysmal thrombus and prevents both its displacement into the parent artery and its fragmentation. It is possible to introduce, deliver, and detach more than one coil in the aneurysm, depending on the size of the lesion. In 18 small aneurysms (<12 mm) an average of 68 cm of platinum coils were detached. This length increases for larger aneurysms.

After detachment of the first coil, it is no longer possible to see under fluoroscopy the distal intra-aneurysmal marker of the microcatheter and the platinum–stainless-steel junction of the subsequent coil(s) emerging from the microcatheter. This is because the radiopaque intra-aneurysmal platinum covers the microcatheter tip, preventing the correct visualization of the platinum–stainless-steel junction. The visualization of the proximal radiopaque markers located on the microcatheter (3 cm before its tip) and on the detachable coils (2.8 cm before the junction) allow a safe delivery of the coils subsequent to the first one: When the two markers (inside the parent artery and 3 cm proximal to the aneurysm) match, it means that the platinum–stainless-steel junction is 2–3 mm beyond the microcatheter tip—that is, in the correct position to be detached. It is imperative that the platinum–stainless-steel junction emerges from the microcatheter for no more than 2–3 mm. In fact, the stainless steel coil proximal to the platinum portion is relatively stiff and might perforate the aneurysm if pushed against its wall.

An angiogram is performed after the detachment of each coil in order to assess the amount of occlusion achieved and to make a decision about whether to use more coils or not. At the end of the embolization proce-

dure, the microcatheter is slowly removed from the aneurysm. A postembolization angiogram is obtained to assess the final result, the proper placement of the detached coil(s), and the patency of the parent artery and adjacent vessels. Then, heparinization is reversed using protamine sulfate. The patient is hospitalized for 3 days. Follow-up angiography is performed at 1 week, 3 months, and 12 months.

## Results

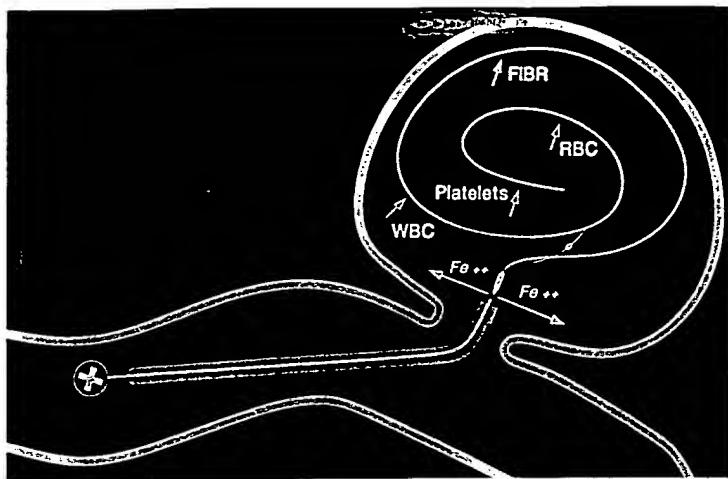
Table 1 summarizes the treatment, complications, results, and outcome in a series of 39 patients. It was always possible to enter all aneurysms with a microcatheter-guidewire combination and to retrieve the coil(s) from the aneurysm if inappropriate in size or position. Failure of electrolytic detachment never occurred. Intra-aneurysmal thrombosis ranging from 70% to 100% was achieved in all cases. Incomplete aneurysm occlusion resulted from partial coil filling of the aneurysm. The portions of the aneurysm filled with coils underwent thrombosis in 100% of cases. The immediate post-thrombosis angiogram demonstrated partial thrombosis and occlusion of aneurysms not packed with enough coils. Nevertheless, progressive thrombosis takes place, within the mesh of coils, in the hours that follow the procedure.

In this series there was one transient neurological deficit (aphasia in case 5, completely regressed in 2 days) and one permanent neurological deficit (hemianopia in case 21). The three patients who were originally in grade V (cases 19, 23, and 29) and one patient who was in grade IV (case 35) died.

In this series of 39 patients treated with endovascular electrothrombosis by means of detachable coils, all aneurysms were considered to be difficult and at high risk for surgery. Balloon embolization was not used because it is believed that detachable coils are less traumatic than balloons in that endovascular electrothrombosis carries less risk of rupturing an aneurysm, especially in the acute phase after SAH. Furthermore, in four instances (cases 4, 5, 13, and 25), balloon embolization would have led to occlusion of normal vessels arising from the aneurysmal base.

In this preliminary clinical experience, we observed one transient (case 5) and one permanent (case 21: hemianopia) neurological deficit after embolization. Four patients (cases 19, 23, 29, and 35) who were devastated by the original bleeding eventually died.

It has been possible to achieve complete aneurysm occlusion in seven cases (cases 15, 19, 21, 22, 24, 29, and 33) and satisfactory aneurysm occlusion in nine more cases (cases 6, 11, 14, 17, 26, 27, 28, 31, and 32). In the remaining cases the aneurysmal neck was too wide (and/or normal arteries were arising from aneurysmal sac) to achieve complete aneurysm occlusion. It is possible to say that a complete aneurysm occlusion can be achieved



**FIG. 7.** Diagrammatical representation of the effects of electric current in an aneurysm. The positively charged intra-aneurysmal platinum attracts the negatively charged red blood cells (RBC), white blood cells (WBC), platelets, and fibrinogen (FIBR), and this initiates thrombus formation. At the same time, the electric current induces migration of ferrous ions from the stainless steel, thus dissolving it and detaching the platinum component within the aneurysm.

in small neck aneurysms (Figs. 8 and 9). Achieving the same results is more difficult in wide neck aneurysms because of the potential danger of coil migration into the parent vessel.

Detachable coils are very soft; they adapt to the shape of the aneurysm without significant increase in intra-aneurysmal pressure. In addition, the risk of rupturing the aneurysm seems to be low. In six instances (cases 3, 16, 19, 23, 29, and 33) the aneurysms were treated in the acute post-hemorrhagic phase—that is, within 48 hr after SAH (Fig. 9).

It is possible to occlude aneurysms in more than one session (cases 10 and 14), thus progressively embolizing the aneurysmal sac. In mostly thrombosed aneurysms, coils do not seem to migrate inside the clot surrounding the residual lumen of the aneurysm (cases 8, 14, and 27 had repeated angiograms showing no coil migration).

Intra-aneurysmal thrombosis progresses with time. It is believed that this is due to two factors: (i) In the hours that follow embolization, more blood components are

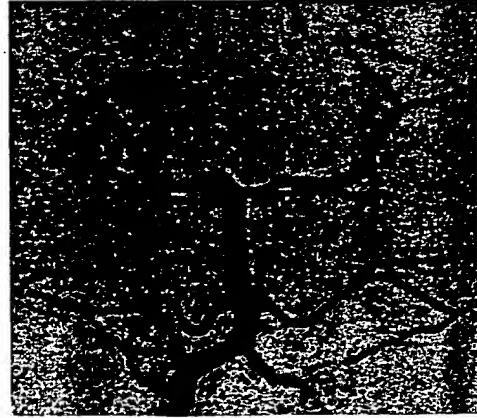
entrapped within the network of coils; (ii) during the procedure, systemic heparinization impedes intra-aneurysmal clot formation; as soon as heparin is reversed, clot formation within the coils is enhanced. It is believed that, aside from producing electrothrombosis, the coils are a packing material that holds the intra-aneurysmal thrombus and prevents both its displacement into the parent artery and its fragmentation. This prevents distal embolization.

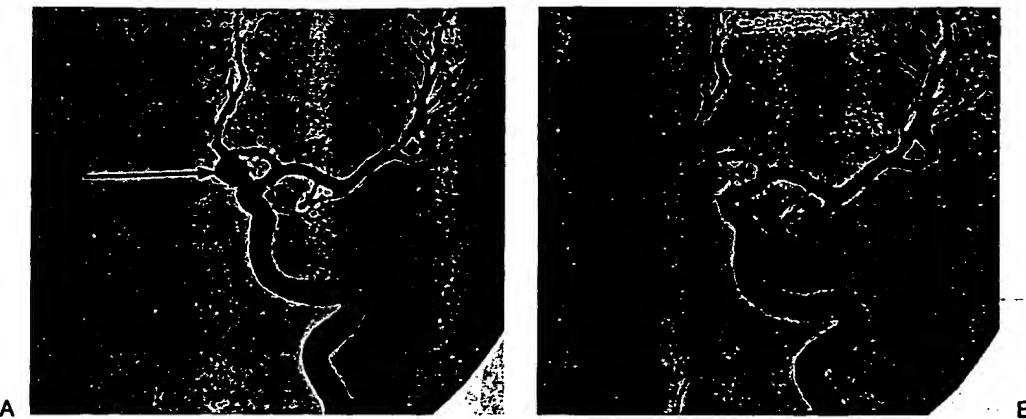
In all cases but two (cases 9 and 18), the parent vessel has been preserved. This result was achieved mainly because it is possible to retrieve the coil into the microcatheter if a satisfactory position in the aneurysm has not been reached. It is then possible to try again with the same coil, or with a different-sized one, to achieve a better intra-aneurysmal coil position.

In two cases (cases 3 and 16) the aneurysms were surgically clipped (3 months after occlusion of the dome and of most of the sac of the aneurysms by means of detachable coils).



**FIG. 8.** A: Right vertebral angiogram, anteroposterior view, showing a basilar bifurcation aneurysm (arrow). The aneurysm had ruptured 5 days before endovascular treatment (case 35). B: After endovascular deposit of seven platinum detachable coils for a total length of 93 cm, the aneurysm is occluded while the normal arteries have been preserved.





**FIG. 9.** A: Left internal carotid angiogram performed on a pregnant woman showing a carotid ophthalmic aneurysm (arrow). The aneurysm had ruptured 48 hr before endovascular treatment (case 33). B: Six detachable coils, for a total length of 68 cm, were delivered and detached by electrolysis into the aneurysm, thus occluding its sac and neck. The immediate postembolization left internal carotid angiogram is shown.

A major goal in the management of ruptured intracranial aneurysms is to treat the patient as soon as possible after his admission in the hospital, possibly within 2 days after SAH. The recently published results of the International Cooperative Study in Timing of Aneurysm Surgery (2,3) confirm that an early surgery (1–3 days after SAH) carries more operative risks than does a delayed intervention. A delayed intervention carries lower morbidity and mortality rates. On the other hand, many patients die while waiting for the best surgical timing (subsequent to day 10 after SAH) because of rebleeding. With this device, implantable at the time of the diagnostic angiogram, it may be possible to avoid early rebleeding.

This technique appears to be promising for the therapeutic management of high-risk intracranial saccular aneurysms. The suppleness and flexibility of the platinum coils allow the occlusion of an aneurysm without deforming its original shape; thus this technique may be applied in the acute phase of SAH, when the aneurysm is still "hot."

In this preliminary experience with 39 patients, the follow-up period is short. This is because the technique has been applied only recently in the clinical setting (March 6, 1990).

Long-term angiographic and clinical follow-up are necessary to achieve an accurate idea about advantages and limitations of this newly developed aneurysm occlusion device.

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